PATENT 1510-1121

IN THE U.S. PATENT AND TRADEMARK OFFICE

Applicants: Lars NILSSON et al.

Confirmation: 4917

Serial No.: 11/527,523 10/593639

Art Unit:

1632

Filed:

September 21, 2006

Examiner:

Not assigned

For:

TRANSGENIC MODEL FOR ALZHEIMER'S DISEASE

INFORMATION DISCLOSURE STATEMENT

Assistant Commissioner for Patents

December 29, 2006

P.O. Box 1450

Alexandria, VA 22313-1450

Sir:

In compliance with Rules 1.97 and 1.98, and in fulfillment of the duty of disclosure under Rule 1.56, included with the attached Form PTO-1449 are copies of the listed references, which are hereby made of record in the above-identified application.

This Information Disclosure Statement is being submitted prior to issuance of an action on the merits; therefore, no fee is required.

The Examiner is courteously requested to initial and return a copy of the accompanying Form PTO-1449 to confirm entry into the record and consideration of the listed references.

Respectfully submitted,

YOUNG & THOMPSON

BY:

Robert J. Patch Reg. No. 17,355

745 South 23rd Street Arlington, VA 22202 703-521-2297 (telephone) 703-685-0573 (telecopier I) 703-979-4709 (telecopier II)

RJP:rk .

INFORMATION DISCLOSURE CITATION
IN AN APPLICATION

Attorney Docket No.: 1510-1121

Application No.: 10/593.639

Applicant:

Lars NILSSON et al.

Filing Date:

Group Art Unit: 1632

(Use several sheets if necessary) **September 21, 2006**

U.S. PATENT DOCUMENTS

Examiner Initial	Document Number	Date	Name	Class	Subclass	Filing date (if appropriate)		
EODEICH DATENT DOCUMENTO								

REIGN PATENT DOCUMENTS

Examiner Initial	Document Number	Date	Country	Class	Subclass	Translation	
						Yes	No
	WO 02/003911	01-17-2002	WIPO				
	WO 02/102412	12-27-2002	WIPO				
	WO 04/041213	05-21-2004	WIPO				

OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)

CAI et al., "Release of excess amyloid b protein from a mutant amyloid b protein precursor," Science, Vol. 259, No. 5094, January 22, 1993, pp. 514-516.

CAIRNS et al., "BA4 protein deposition in familial Alzheimer's disease with the mutation in codon 717 of the BA4 amyloid precursor protein gene and sporadic Alzheimer's disease," Neuroscience Letters, Vol. 149, 1993, pp. 137-140.

CHARTIER-HARLIN et al., "Letters to Nature: Early-onset Alzheimer's disease caused by mutations at codon 717 of the β-amyloid precursor protein gene," Nature, Vol. 353, October 31, 1991, pp. 844-846.

CHISHTI et al., "Early-onset amyloid deposition and cognitive deficits in transgenic mice expressing a double mutant form of amyloid precursor protein 695," The Journal of Biological Chemistry, Vol. 276, No. 24, June 15, 2001, pp. 21562-21570.

CHUI et al., "Transgenic mice with Alzheimer presenilin 1 mutations show accelerated neurodegeneration without amyloid plaque formation," Nature Medicine, Vol. 5, No. 5, May 1999, pp. 560-564.

CITRON et al., "Letters to Nature: Mutation of the B-amyloid precursor protein in familial Alzheimer's disease increases B-protein production," Nature, Vol. 360, December 17, 1992, pp. 672-674.

CORDER et al., "Gene dose of apoliprotein E type 4 allele and the risk of Alzheimer's disease in late onset families," Science, Vol. 261, August 13, 1993, pp. 921-923.

CROWTHER et al., "Intraneuronal A B, non-amyloid aggregates and neurodegeneration in a drosophila model of Alzheimer's disease," Neuroscience, Vol. 132, 2005, pp. 123-135

DEMATTOS et al., "Reports: Brain to plasma amyloid- B efflux: A measure of brain amyloid burden in a mouse model of Alzheimer's disease," Science, Vol. 295, March 22, 2002, pp. 2264-2267

EDBAUER et al., "Reconstitution of γ-secretase activity," Nature Cell Biology, Vol. 5, May 2003, pp. 486-488. FAGAN et al., "Human and murine ApoE markedly alters A B metabolism before and after plaque formation in a mouse model of Alzheimer's disease," Neurobiology of Disease, Vol. 9, 2002 pp. 305-318.

GAMES et al., "Letters to Nature: Alzheimer-type neuropathology in transgenic mice overexpressing V717F β-amyloid precursor protein," Nature, Vol. 373, February 9, 1995, pp. 523-527.

GLENNER et al., "Alzheimer's disease: Initial report of the purification and characterization of a novel cerebrovascular amyloid protein," Biochemical and Biophysical Research Communications," Vol. 120, No. 3, May 18, 1984, pp. 885-890.

GOATE et al., "Letters to Nature: Segregation of a missense mutation in the amyloid precursor protein gene with familiar Alzheimer's disease," Nature, Vol. 349, February 21, 1991, pp. 704-705.

HOLCOMB et al., "Accelerated Alzheimer-type phenotype in transgenic mice carrying both mutant amyloid precursor protein and presenilin 1 transgenes," Nature Medicine, Vol. 4, No. 1, January 1998, pp. 97-100.

HSIAO et al., "Correlative Memory Deficits, Ab Elevation, and amyloid plaques in transgenic mice," Science,

New Series, Vol. 274, No. 5284, October 4, 1996, pp. 99-102.

IWATA et al., "Clearance of amyloid-β peptide in the brain by adeno-associated viral vector-mediated neprilysin gene transfer," 33" Annual Meeting of the Society of Neuroscience, New Orleans, November 8-12, 2003, 2 pp. abstract.

EXAMINER:

DATE CONSIDERED

EXAMINER: Initial if citation considered, whether or not citation is in conformance with MPEP § 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to the applicant.

FORM PTO-1449 Sheet <u>2</u> of <u>2</u>								
INFORMATION DISCLOSURE CITATION	Attorney Docket No.: 1510-1121	Application No.: 10/593,639						
IN AN APPLICATION	Applicant: Lars NILSSON et al.							
(Use several sheets if necessary)	Filing Date: September 21, 2006	Group Art Unit: 1632						
OTHER DOCUMENTS (Including A	uthor, Title, Date, Pertinent Page	s, Etc.)						
surface receptor," Nature, Vol. 325, February 19	KANG et al., "Letters to <i>Nature</i> : The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor," <i>Nature</i> , Vol. 325, February 19, 1987, pp. 733-736.							
European Journal of Neuroscience, Vol. 19, 200	KLYUBIN et al., "Soluble arctic amyloid B protein inhibits hippocampal long-term potentiation in vitro," European Journal of Neuroscience, Vol. 19, 2004, pp. 2839-2846.							
LANTOS et al., "Familial Alzheimer's disease sporadic Alzheimer's disease have the same cpp. 221-224.	LANTOS et al., "Familial Alzheimer's disease with the amyloid precursor protein position 717 mutation and sporadic Alzheimer's disease have the same cytoskeletal pathology," Neuroscience Letters, Vol. 137, 1992,							
protofibrils, including amyloid pores," Journal of	LASHUEL et al., "Mixtures of wild-type and a pathogenic (E22G) form of A B40 <i>in vitro</i> accumulate protofibrils, including amyloid pores," <i>Journal of Molecular Biology</i> , 2003, Vol. 332, pp. 795-808.							
precursor protein in the hippocampus of ac 2479-2487.	LI et al., "Intracellular accumulation of detergent-soluble amyloidogenic A B fragment of Alzheimer's disease precursor protein in the hippocampus of aged transgenic mice," <i>Journal of Neurobiology</i> , 1999, pp.							
LORENZO et al., "B-amyloid neurotoxicity Proceedings of the National Academy of Science	e USA," Vol. 91, December 1994, _I	op. 12243-12247.						
	MASTERS et al., "Amyloid plaque core protein in Alzheimer disease and Down Syndrome," <i>Proceedings of the National Academy of Sciences USA</i> , Vol. 82, June 1985, pp. 4245-4249.							
MULLAN et al., "A pathogenic mutation for pro	MULLAN et al., "A pathogenic mutation for probable Alzheimer's disease in the APP gene at the N-terminus of β-amyloid," <i>Nature Genetics</i> , Vol. 1, August 1992, pp. 345-347.							
MURRELL et al., "A mutation in the amyloid	d precursor protein associated w	vith hereditary Alzheimer's						
NASLUND et al., "Correlation between eleva	disease," Science, Vol. 254, No. 5028, October 4, 1991, pp. 97-99. NASLUND et al., "Correlation between elevated levels of amyloid β-peptide in the brain and cognitive decline." Journal of the American Medical Association, Vol. 383, No. 13, Morch 23/39, 2009, pp. 1571, 1577							
NILSBERTH et al., "The 'Arctic' APP mutati	decline," Journal of the American Medical Association, Vol. 283, No. 12, March 22/29, 2000, pp. 1571-1577. NILSBERTH et al., "The 'Arctic' APP mutation (E693G) causes Alzheimer's disease by enhanced Aβ							
NILSSON et al., "α-1-Antichymotrypsin promot	protofibril formation," <i>Neuroscience</i> , Vol. 4, No. 9, September 2001, pp. 887-893. NILSSON et al., "α-1-Antichymotrypsin promotes β-sheet amyloid plaque deposition in a transgenic mouse							
PIKE et al., "In vitro aging of β-amyloid pro	model of Alzheimer's disease," <i>The Journal of Neurosciences</i> , Vol. 21, No. 5, March 1, 2001, pp. 1444-1451. PIKE et al., "In vitro aging of β-amyloid protein causes peptide aggregation and neurotoxicity," <i>Brain</i>							
Research, Vol. 563, 1991, pp. 311-314. ROHER et al., "The human amyloid-β precurse	or protein ₇₇₀ mutation V717F gene	rates peptides longer than						
amyloid-β-(40-42) and flocculent amyloid aggree February 13, 2004, pp. 5829-5836.	•	• , , , ,						
increased in vivo by the presentlin 1 and 2 and	SCHEUNER et al., "Secreted amyloid β-protein similar to that in the senile plaques of Alzheimer's disease is increased <i>in vivo</i> by the presentiin 1 and 2 and <i>APP</i> mutations linked to familial Alzheimer's disease," <i>Nature Medicine</i> , Vol. 2, No. 8, August 1996, pp. 864-870.							
	SELKOE, "Cell biology of the β-amyloid protein precursor and the mechanism of Alzheimer's disease," <i>Annual Review of Cell Biology</i> , Vol. 10, 1994, pp. 373-403.							
	SELKOE, "Normal and abnormal biology of the β-amyloid precursor protein," Annual Review of							
	STENH et al., "The Arctic mutation interferes with processing of the amyloid protein precursor," NeuroReport,							
STRITTMATTER et al., "Apoliprotein E: High-a allele in late-onset familial Alzheimer disease,"	STRITTMATTER et al., "Apoliprotein E: High-avidity binding to β-amyloid and increased frequency of Type 4 allele in late-onset familial Alzheimer disease," <i>Proceedings of the National Academy of Science USA</i> ," Vol.							
90, March 1993, pp. 1977-1981. STURCHLER-PIERRAT et al., "Two amyloid	90, March 1993, pp. 1977-1981. STURCHLER-PIERRAT et al., "Two amyloid precursor protein transgenic mouse models with Alzheimer							
disease-like pathology," <i>Proceedings of the N</i> November 1997, pp. 13287-13292.	lational Academy of Science US	A," Neurobiology, Vol. 94,						
SUZUKI et al., "An increased percentage of lo precursor (baPP\$_(717)\$) Mutants," Science, V	ol. 264, No. 5163, May 27, 1994, p	p. 1336-1340.						
WALSH et al., "Letters to Nature: Naturally	WALSH et al., "Letters to Nature: Naturally secreted oligomers of amyloid β protein potently inhibit hippocampal long-term potentiation in vivo," <i>Nature</i> , Vol. 416, April 4, 2002, pp. 535-539.							

EXAMINER:

DATE CONSIDERED

EXAMINER: Initial if citation considered, whether or not citation is in conformance with MPEP § 609. Draw line through citation if not in conformance and not considered. Include copy of this form with next communication to the applicant.